Results: 15 randomized trials randomizing 1054 patients were analyzed. Naloxone had significant effect on improvement of hepatic encephalopathy (relative risk 1.46; 95 percent CI 1.27 to 1.67; P = 0.0005). This comparison showed significantly statistical heterogeneity (P = 0.10, and χ^2= 44.93). Subgroup analysis showed that naloxone administered both by injected and infusion route could be effective (relative risk 1.34; 95 percent CI 1.17 to 1.53; P = 0.0001) and there was significant effect in trials by infusion route (relative risk 1.42; 95 percent CI 1.19 to 1.69; P = 0.0001). Adverse effects were observed in only 8 patients treated with naloxone.
Conclusion: Naloxone could get patients with hepatic encephalopathy improved, however because of the studies were of generally poor quality, we are unable to make firm conclusions. It is possible that further investigation in well-designed trials may help confirm our results.
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PP-048 Autoimmune hepatitis complicated with Sjögren's syndrome: a case report

Ying-Qiong Peng[1], Meng-Hou Lu*[,1,2]. *[1]Department of Infectious Diseases, Xiangya Hospital, Central-South University, Xiangya Road, Changsha, Hunan, PR China; [2]Department of Infectious Diseases, Xiangya Hospital, Central-South University*

Introduction: Sjogren syndrome (SS) is a chronic autoimmune disorder that may affects salivary, lachrymal glands, liver, blood, joints and others with diverse clinical manifestations. We presented a case of autoimmune hepatitis complicated with SS, which mainly presented recurrent abnormal liver enzyme tests of unknown origin.
Case description: A 45-year-old woman had a 5-month history of recurrent weakness and anorexia, and 15-day history of jaundice. Physical examination revealed moderate jaundice. Laboratory findings revealed hepatic dysfunction, including high level aspartate aminotransferase 999.1 IU/L, alanine aminotransferase 597.8 IU/L, direct bilirubin 51.8 mg/dl, and total bilirubin 95.2 mg/dl. The titer of anti-nuclear antibody is 1:320. Multiple antibodies were negative, including anti-hepatitis A, B, C, E viruses, Anti-double chain DNA, mitochondrial antibody, smooth-muscle autoantibody, anti-neutrophil cytoplasmic antibody, and anti-SSA/B antibody. Histologic examination of liver showed hydropic degeneration of some hepatocytes. Inflammatory cells were observed in portal areas. Biopsy of salivary labial gland demonstrated sialoadenitis. Positive fluorescent stain of the right cornea. Those confirmed the diagnosis of autoimmune hepatitis complicated with SS.
SS is not uncommon. Patients with hyperglobulinemias, rampant caries, ophthalmos purulent discharge, recurrent parotid enlargement, joint pain, recurrent abnormal liver enzyme or jaundice of unknown origin, and all suspected virual hepatitis should be asked about the history of oral or eye dryness. If necessary, anti-nuclear, anti-SSA/B antibody, salivary gland biopsy, corneal fluorescent exam, and parotid imagery should be obtained to minimize misdiagnosis.

PP-049 The immunostaining of programmed death 1 (PD-1) and its ligands in liver tissues of patients with hepatitis and hepatocellular carcinoma

Baoju Wang*[,1], Junjie Bao[1,2], Min Jiang[3], Junzhong Wang[1], Yang Wang[1], Mingyou Xing[4], Wanguang Zhang[5], Junying Qi[4], Michael Roggendorf[6], Mengji Lu[6], Dongliang Yang[1]. *[1]Division of Clinical Immunology, Tongji Hospital of Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China; [2]Guangzhou Women and Children's Medical Center, Guangzhou, China; [3]Department of Microbiology, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China; [4]Department of Infectious Disease, Tongji Hospital of Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China; [5]Hepatic Surgery Center, Tongji Hospital of Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China; [6]Institut für Virologie, Universitätsklinkum Essen, Essen, Germany*

Goals: The programmed death 1 (PD-1) and its ligands (PD-L1 and PD-L2) are known as a system for negative regulation of T-cell activation. Recent investigations indicated the PD-1/PD-Ls system plays a significant role in persistent viral infection and tumor development. Thus, it is warrant to investigate the expression of PD-1, PD-L1 and -2 in liver tissues in the context of chronic hepatitis and those of hepatocellular carcinomas (HCC) and evaluated their relationships with clinical and pathological variables.
Study: Liver biopsies and HCC specimens from patients were collected and histologically examined. The expression of PD-1, PD-L1, and PD-L2 in biopsy specimens of chronic hepatitis and of HCC specimens was evaluated by immunohistochemical staining.
Results: The expression of PD-1 was found in liver infiltrated lymphocytes. In the contrast, PD-L1 and -L2 were expressed in Kupffer cells, liver sinoidal endothetial cells (LSECs), and tumor cells. The expression of PD-L1 was significantly correlated with HBV infection and with the stage of HCC. PD-1 and PD-Ls were significantly up-regulated in HCC specimens.
Conclusions: The intrahepatic expression of PD-L1 was significantly up-regulated in HBV infection, suggesting that the PD-L1 may contribute to negative regulation of immune response in chronic hepatitis B. It is worthy to examine whether the high level expression of PD-1 and PD-Ls in HCC specimens play a role in the carcinogenesis and immune evasion of tumors.

PP-050 Alpha-fetoprotein induced phosphorylation of AKT via inhibited the activity of PTEN in human hepatoma Bel 7402 cells

Mengsen Li*[,1,2], Sheng Zhou[1], Mingyue Zhu[1], Xiaoying Wang[1], Xinhua Liu[2], Gang Li[2]. *[1]Key Laboratory of Molecular Biology, Hainan Medical College; [2]Department of Biochemistry and Molecular Biology, Peking University Health Science Center*

Objective: The events of hepetocellular cancinoma cells (HCC) correlated to infection of hepatitis virus. PTEN/AKT signal pathway plays a central role in the proliferation of the tumor cells. The present investigation purpose to explore alpha-fetoprotein (AFP) influences on the transduction of PTEN/AKT signal pathway in HCC.
Methods: Western Blotting was utilized to detecting the expression of PTEN and the phosphorylation of protein kinase B (AKT) in Bel 7402 cells. AFP interacted with PTEN was analyzed by co-immunoprecipitation (Co-IP); Laser confocal microscopy was used to observe co-localization of AFP with PTEN and the fluorescence resonance energy transfer (FRET) of FITC (labeled PTEN) and TRITC (labeled AFP); Short small RNA interfering (RNAi) was applied to block the expression of AFP.
Results: All *trans* retinoic acid (ATRA) (160μmol/L) mildly promoted the expression of PTEN in Bel 7402 cells; Co-IP indicated that AFP could interact with PTEN, co-localization of PTEN and